Elevated Intra-abdominal Pressure and Renal Function

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The effect of increased intra-abdominal pressure on cardiac output and renal function was investigated using anesthetized dogs into whom inflatable intraperitoneal bags were placed. Hemodynamic and renal function measurements were made at intra-abdominal pressures of 0, 20, and 40 mmHg. Renal blood flow and glomerular filtration rate decreased to less than 25% of normal when the intra-abdominal pressure was elevated to 20 mmHg. At 40 mmHg intra-abdominal pressure, three dogs became anuric, and the renal blood flow and glomerular filtration rate of the remaining dogs was 7% of normal, while cardiac output was reduced to 37% of normal. Expansion of the blood volume using Dextran-40 easily corrected the deficit in cardiac output, but renal blood flow and glomerular filtration rate remained less than 25% of normal. Renal vascular resistance increased 555% when the intra-abdominal pressure was elevated from 0 to 20 mmHg, an increase fifteen-fold that of systemic vascular resistance. This suggests that the impairment in renal function produced by increased intra-abdominal pressure is a local phenomenon caused by direct renal compression and is not related to cardiac output.

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INTRA-ABDOMINAL PRESSURE can be increased by intestinal obstruction, ascites, ruptured abdominal aortic aneurysm, postoperative bleeding, and the application of external counterpressure suits (military antishock trousers, MAST). The respiratory and cardiovascular derangements produced by increased intra-abdominal pressure are well described.[1-4] Less attention has been focused on renal function in this situation, but most investigators have observed a decreasing urinary output with increasing intra-abdominal pressure.[5,6] It has been assumed that this decreased urinary output is due to a decreased cardiac ouput, which itself results from decreased venous return. If this theory is correct, then expansion of the blood volume to increase cardiac output should improve urine production. The present study was designed to test this hypothesis and to define further the effect of elevated intra-abdominal pressure on renal function.

Methods

Seven mongrel dogs were anesthetized with pentobarbitol (25 mg/kg), intubated with a cuffed endotracheal tube, and placed on volume-cycled ventilators at a tidal volume of 15 ml/kg. Adjustment of the rate of ventilation maintained arterial blood gases and pH within normal limits. Polyethylene catheters were inserted into the right carotid and femoral arteries, left femoral vein, abdominal aorta, and inferior vena cava. From a right femoral cutdown, stiff plastic catheters were introduced into the left renal artery and vein. Superior vena caval and pulmonary artery pressures were measured through a no. 7 French Swan–Ganz pulmonary artery catheter. An indwelling transurethral catheter was placed in the bladder of all the dogs, and in two dogs, stiff, hollow ureteral stents were delivered into the renal pelvis bilaterally. A midline laparotomy was performed, the splenic hilum was ligated, the positions of the catheters in the renal artery and vein were confirmed, and an inflatable bag was placed in the peritoneal cavity. The abdominal wall was closed in two layers. When the arterial blood and gases and pH were normal and the urine output was 1 to 2 ml/kg/hr, an initial dose and then a continuous infusion (at an infusion rate of 1 ml/min) of tritiated para-aminohippuric acid and carbon-14 inulin in normal saline were administered to produce a steady state concentration of radionuclides in the serum. Baseline determinations of glomerular filtration rate and renal blood flow were calculated from urine volume and from serum and urine concentrations of inulin and para-aminohippuric acid. Radionuclide counts were performed using a liquid scintillation spectrometer. Additional baseline determinations included a hematocrit, arterial blood gases and pH, pulse rate, cardiac output (by the thermodilution technique), and measurement of carotid, renal, femoral, and pulmonary artery, pulmonary artery wedge, renal and femoral vein, and superior and inferior vena caval pressures. The baseline hemodynamic and renal function measurements were repeated at 15-minute intervals until a steady state was reached. Then, intra-abdominal pressure was elevated to 20 mmHg by air insufflation of the intraperitoneal

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Submitted for publication: March 23, 1982.

0003-4932/82/1100/0594 $01.00

bag. After 20 minutes, two hemodynamic and renal function measurements were taken, 15 minutes apart. Then intra-abdominal pressure was elevated to 40 mmHg, and after 20 minutes, the same two measurements were made, 15 minutes apart. After 60 minutes at 40 mmHg intra-abdominal pressure, the intravascular volume was expanded by the serial intravenous administration of Dextran-40 in doses of 1 ml/kg, until the cardiac output was increased to twice the baseline cardiac output. Again, after 20 minutes, two hemodynamic and renal function measurements were made 15 minutes apart. The intraperitoneal bag was then deflated, and three additional sets of measurements were made at 15-minute intervals.

Results

The mean glomerular filtration rate of seven dogs decreased from a baseline value of 46 ± 4.0 ml/min to 10.2 ± 2.6 ml/min at 20 mmHg intra-abdominal pressure (Table 1). At 40 mmHg intra-abdominal pressure, three dogs became anuric, and the glomerular filtration rate in the remaining four dogs was reduced to 3.3 ± 0.8 ml/min. Renal blood flow paralleled this trend. The renal response to increased intra-abdominal pressure was similar in the two dogs with and the five dogs without ureteral stents.

The mean baseline cardiac output for all seven dogs was 2.14 ± 0.2 liters/min and was reduced to 1.78 ± 0.3 liters/min at 20 mmHg intra-abdominal pressure. It was further reduced to 0.8 ± 0.6 liters/min at 40 mmHg intra-abdominal pressure (Table 2). Blood volume augmentation with Dextran-40 produced a mean cardiac output twice that calculated for the baseline; however, the glomerular filtration rate and renal blood flow remained markedly impaired.

Raising intra-abdominal pressure from 0 to 20 mmHg increased renal vascular resistance 555% ± 203%, fifteenfold greater than the 30.4% ± 17.0% increase of systemic vascular resistance. At 40 mmHg intra-abdominal pressure, three of the dogs became anuric, and the renal vascular resistance could not be calculated. The renal vascular resistance of the remaining four dogs was 80,936 ± 18,280 dynes-sec/cm^5, 1,512% greater than resistance at the baseline. Mean systemic vascular resistance for the same four dogs was 7,531 ± 824 dynes-sec/cm^5 at 40 mmHg intra-abdominal pressure (Table 3).

Arterial and venous pressures are listed in Table 4. Inferior vena caval pressure always equalled intra-abdominal pressure, and renal vein pressure always exceeded intra-abdominal pressure by 1 to 2 mmHg. Pulmonary artery wedge pressure increased slightly with increasing intra-abdominal pressure, but this increase was not statistically significant. The mean hematocrit was 41% at the start of the study and 35.4% at the completion, reflecting the dilutional effect of the blood volume expansion with Dextran-40.

TABLE 1. *Renal Function*

Intra-abdominal Pressure (mmHg)	Renal Blood Flow (cc/min)	Glomerular Filtration Rate (cc/min)
0	160.0 ± 17	46.0 ± 4.0
20	36.0 ± 12*	10.2 ± 2.6*
40	9.2 ± 3.4*#	3.3 ± 0.8*#
40 (after blood volume expansion)	31.3 ± 11*	9.3 ± 3.1*
0 (after deflation of intra-abdominal bag)	94.0 ± 17*	34.1 ± 13.3*
0 (#2)	117.0 ± 19	43.2 ± 15.3
0 (#3)	99.0 ± 15	35.5 ± 11.2

± = standard error of the mean.
* = $p < .05$.
# = 3 dogs anuric, N = 4.

TABLE 2. *Cardiac Function*

Intra-abdominal Pressure (mmHg)	Pulse Rate (beats/min)	Cardiac Output (liters/min)
0	157 ± 9	2.14 ± 0.2
20	147 ± 6	1.78 ± 0.3*
40	153 ± 16	0.8 ± 0.6*
40 (after blood volume expansion)	143 ± 11	4.3 ± 0.8*
0 (after deflation of intra-abdominal bag)	138 ± 11	7.0 ± 1.3*

± = standard error of the mean.
* = $p < .05$.

Discussion

This study demonstrates that intra-abdominal pressure of as little as 20 mmHg markedly impairs renal function, reducing glomerular filtration rate and renal blood flow to 21% and 23% of their baseline values, respectively. Increasing intra-abdominal pressure to 40 mmHg caused three dogs to become anuric; consequently, their renal blood flow could not be determined. Glomerular filtration rate and renal blood flow were

TABLE 3. *Vascular Resistances*

Intra-abdominal Pressure (mmHg)	Systemic Vascular Resistance (dynes-sec/cm^5)	Renal Vascular Resistance (dynes-sec/cm^5)
0	4474 ± 372	5,350 ± 431
20	5834 ± 1528	29,674 ± 12,517*
40	7531 ± 824*#	80,936 ± 18,280*#

± = standard error of the mean.
* = $p < .05$, significant change from baseline determination.
# = N = 4, three dogs anuric.

TABLE 4. *Intravascular Pressures (mmHg)*

Intra-abdominal	Carotid Artery	Renal Artery	Renal Vein	Inferior Vena Cava	Superior Vena Cava	Pulmonary Artery Occluded
0	109 ± 9	109 ± 8	3.1 ± 0.9	2.6 ± 0.7	1.6 ± 0.4	2.0 ± 0.4
20	110 ± 12	110 ± 11	21.0 ± 0.8*	20.0 ± 0.7*	2.5 ± 0.9	2.5 ± 0.9
40	94 ± 8	95 ± 9	41.0 ± 0.8*	39.4 ± 0.6*	2.1 ± 0.6	4.4 ± 1.9
40 (after blood volume expansion	120 ± 4	119 ± 5	41.2 ± 0.6*	39.5 ± 1.0*	4.1 ± 0.9*	8.2 ± 2.3*
0 (after deflation of intra-abdominal bag)	117 ± 8	117 ± 8	4.1 ± 0.8	3.4 ± 0.5	3.0 ± 0.4*	4.1 ± 0.5*

± = standard error of the mean.

* = $p < .5$, significant difference from baseline.

only 7% of normal in the four dogs who continued to produce urine at this level. Becket demonstrated similar trends in goat and calf models in which ruminal insufflation was employed to elevate intra-abdominal pressure.[5] Shenasky and Gillenwater observed a comparable impairment of urine production in a dog model subjected to constant external counterpressure by the use of an antigravity suit.[6]

These results reaffirm the findings of Kashtan, Trinkle, and Shenasky, who, in three separate studies, demonstrated that increased intra-abdominal pressure decreased cardiac output in normovolemic dogs.[2,3,6] Most investigators have found some degree of increase in systemic vascular resistance with increased intra-abdominal pressure, and they imply that this contributes to the decrease in cardiac output. These findings are in agreement. The role of preload in this situation is more controversial. Richardson and Trinkle observed a falling central venous pressure with increasing intra-abdominal pressure in their series of experiments, and implicate this fall as the major cause of the decreased cardiac output.[3] Kashtan constructed venous return curves in his series of experiments and showed that with increased intra-abdominal pressure in normovolemic dogs, venous return to the right heart fell.[2] No significant change was observed in pulmonary artery occluded pressure or in central venous pressure with increasing intra-abdominal pressure to 40 mmHg, indicating that preload was maintained in this model.

A major drawback of previous investigations of renal function in the face of elevated intra-abdominal pressure has been the failure to define the role of ureteral compression. For that reason, in two dogs in this study, stiff, hollow ureteral stents were delivered into the renal pelvis bilaterally to eliminate the effect of ureteral compression. No significant difference in any parameters of hemodynamic or renal function was observed in the dogs with and without ureteral stents, a finding that tends to negate the role of ureteral compression as a cause of renal dysfunction when the intra-abdominal pressure is elevated to 40 mmHg, and which is consistent with the observation by Vaughan that with acute ureteral obstruction, the renal collecting system can generate pressures up to 90 mmHg.[7] Vaughan also demonstrated that renal blood flow increases acutely with ureteral obstruction. This is opposite to the response the authors observed and further discounts the role of ureteral compression as contributing to renal dysfunction in this model.

Unique to the present study is the observation that the correction of cardiac output did not correct the renal dysfunction. At 40 mmHg intra-abdominal pressure, a mean cardiac output twice that recorded at the baseline was produced with blood volume expansion. The mean glomerular filtration rate remained severely depressed at one fifth of normal. The 555% increase in renal vascular resistance, compared with the 30% increase in systemic vascular resistance at 20 mmHg intra-abdominal pressure, points specifically to a derangement of the kidney. Renal artery pressure is maintained during the incremental increase in intra-abdominal pressure. This suggests that renal parenchymal and renal vein compression are the causes of renal dysfunction when the intra-abdominal pressure is elevated.

Historically, much controversy has existed concerning the normal intra-abdominal pressure in the healthy human.[8] Waggoner, in 1926, demonstrated subatmospheric pressures in the abdominal cavity of normal patients.[9] In 1934, Salkin recorded mean intra-abdominal pressures of 0 to minus 30 mm of water in 88% of 50 normal humans.[10] Overholt recorded, in supine dogs, pressures varying from subatmospheric in the xyphoid area to slightly positive in the dependent reaches of the abdomen.[11] The consensus of research since these early studies, with few exceptions, is that normal mean intra-abdominal pressure is 0 to subatmospheric.[12]

Clearly there are, however, several clinical situations in which intra-abdominal pressure far exceeds this. Pressures of 18 to 80 mmHg have been recorded in patients with cirrhotic ascites.[1,13] Vigorous intestinal peristalsis produces temporary intraluminal pressures of up to 30 mmHg; in the setting of bowel obstruction, constant high pressures are produced.[14] An analogous situation is seen in Beckett's goat model in which he demonstrated

marked renal dysfunction with ruminal insufflation of 20 mmHg, indicating that diffuse intestinal distention raises intra-abdominal pressure sufficiently to compress the kidneys. External counterpressure suits are commonly inflated to 20 to 40 mmHg for autotransfusion in hypovolemic patients and as high as 100 mmHg to control bleeding. Shenasky observed that up to 80% of the inflated pressure of the antigravity suit is transmitted to the retroperitoneum.[4]

Herman and Winton, using a pump-lung-kidney model, were, in 1936, the first to demonstrate that extrarenal pressure as little as 10 mmHg greater than atmospheric pressure impaired renal blood flow and urine production.[15] The authors' findings suggest that a similar response is produced *in vivo* when the kidney of the dog is compressed by an elevated intra-abdominal pressure. The clinical implications are that the renal dysfunction produced by increased intra-abdominal pressure is caused by local compression of the kidney and that poor urine production in the presence of an elevated intra-abdominal pressure should encourage efforts to decompress the peritoneal cavity. These clinical implications apply specifically to patients with postoperative bleeding, which is occasionally left to tamponade, and to patients with tense abdominal distention caused by bowel obstruction who remain oliguric despite adequate fluid resuscitation. Patients with tense ascites represent a more complex situation, but will frequently remain oliguric despite a hyperdynamic circulation. Peritoneojugular shunt decompression of the abdomen often improves urine output in this specific subset of patients, and, although the therapeutic mechanism is not well understood, relief of renal compression may be involved. These findings also suggest that the recent enthusiasm for the use of external counterpressure suits to treat hypovolemic shock,[16] to transport patients with leaking abdominal aortic aneurysms,[17] to tamponade bleeding after closed renal biopsy,[18] and to arrest postoperative intraperitoneal hemorrhage should be tempered by an appreciation for the adverse effect on renal function when the intra-abdominal pressure is elevated in the dog.

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